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REVIEW ARTICLE

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Clinical approach to chronic wound management in older adults

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Abstract

Older adults are at high risk of developing chronic wounds due to numerous changes that occur with aging. It is reasonable to consider chronic wounds as a geriatric syndrome—highly prevalent, multifactorial, and associated with substantial morbidity and mortality. Due to the morbidity and cost associated with chronic wounds, prevention, early diagnosis, and treatment are important. The most common chronic wounds presenting in older adults are pressure and vascular wounds, including those associated with diabetes. Atypical wounds are also common and should raise the suspicion for skin malignancy. Diagnosis is primarily clinical and assessment should include documentation of wound characteristics, such as location, size and depth, presence of slough, drainage, odor, and infection. The mainstay of treatment is based on the TIME principle: Tissue debridement, Infection control, Moisture balance, and optimal wound Edges. The use of protein supplements has been shown to improve wound healing in subsets of older adults. In addition to wound care and optimizing nutrition, disease-specific wound therapy forms an integral part of wound management. Pressure reduction for pressure injury, compression therapy for venous wounds, evaluation of arterial circulation with ABI or arterial Doppler and iCC for diabetic ulcers form the mainstays of therapy. Atypical wounds may present as chronic ulcers and should be biopsied. The goals of treatment should be realistic and for some older adults, palliative wound management may be more appropriate.

KEYWORDS

older adults, wound healing, wounds

BACKGROUND

A chronic wound is defined as one that fails to progress through a timely reparative process to re-establish anatomic and functional skin integrity over 1–3 months.[1,2] The Wound Healing Society categorizes chronic wounds into four different categories based on the causative etiologies: pressure, diabetic, venous, and arterial.

Chronic wounds represent a silent epidemic that affects much of the older population throughout the world.[3] Determining the prevalence of chronic wounds can be challenging due to different categories and overlapping descriptions.[4] According to data from a systematic review published in 2017, chronic wounds affect 5.7 million Americans with an annual cost of $20 billion.[5] The impact is not only economic but significantly affects the quality of life for patients and their families, causing

pain, loss of function, distress, embarrassment, social isolation, hospitalization, or even death.[6]

CHRONIC WOUNDS IN OLDER ADULTS

Older adults are at high risk of developing chronic wounds due to numerous changes that occur with aging. These include a higher prevalence of chronic conditions, such as cardiovascular disease and diabetes, impaired mobility, incontinence, low weight, poor nutritional status, and cognitive impairment. These co-morbidities are often concurrent with additional risk factors including acute exacerbation of illness, multiple medication use, dehydration, and hospitalization.[7] The age-associated changes in the basic biology of the skin also play a significant role in wound formation, chronicity, and healing.[5] Intrinsic changes in skin wound healing that are affected by age include: alterations in the body's inflammatory response, lower levels of supportive extracellular matrix (ECM) and growth factors, delayed epithelialization, and decreased angiogenic activity. All contribute to a lower rate of wound closure in older adults.[8] It is reasonable to consider chronic wounds as one of the geriatric syndromes[9]—highly prevalent, multifactorial, and associated with substantial morbidity and mortality.[10]

Management of chronic wounds should be similar to the approach taken with other geriatric syndromes and encompass a comprehensive and multidisciplinary approach that takes into account many variables. These include changes with age, patient co-morbidities and preferences, medication use, functional and cognitive status, social support, and quality of life.[11]

In this article, we discuss the pathophysiology of chronic wounds and common wounds in older adults, including pressure, vascular and neuropathic wounds. We review their classification, approach to evaluation, and a multidisciplinary approach to management.

PATHOPHYSIOLOGY OF WOUND HEALING

There are four stages of wound healing; hemostasis, inflammation, granulation, and maturation (Figure 1A). The cascade of events begins with clot formation followed by the release of growth factors, chemotaxis, angiogenesis, collagen formation, and epithelialization. Understanding the mechanism and pathophysiology of these stages underlies the foundation for strategies to optimize wound healing. Most wounds heal in 4–6 weeks when they follow a timely reparative process, but ongoing remodeling processes can continue over several months resulting in either maturation or scar formation.

Key points

- Older adults are at high risk of developing chronic wounds.
- Chronic wounds are associated with a high cost and reduced quality of life in older adults.
- The principles of management can and should be modified for a palliative approach to chronic wounds if that is consistent with the older adults' goals of care.

Why Does this Paper Matter?

This article provides the essential knowledge, key concepts, and general principles of chronic wound care in older adults. This will be useful for the reader of this journal in clinical practice, especially in view of the paucity of recent literature about this topic.

Wounds become chronic if they fail to follow a timely reparative process. The exact mechanisms are unknown; however, hypoxia, bacterial colonization, altered cellular responses, and defects in ECM can all contribute to delayed wound healing.[12] Wounds typically arrest during the inflammatory or granulation stages.

The microenvironment of a normal wound is characterized by an abundance of growth factors, well-organized ECM, regulation of proteases (such as matrix metalloproteases [MMPs], tissue inhibitors of metalloproteases [TIMPs], and other modulators of enzyme activity), and a responsive cell population. This is followed by recruitment of endothelial progenitor cells, timely angiogenesis, and proliferation as well as apoptosis of fibroblasts. Normal wounds are also low in bacterial burden.

Chronic wounds, on the other hand, remain in an inflammatory phase, characterized by low levels of growth factors, unregulated protease activity, and high bacterial burden. There is a failure of ECM formation and cell migration, likely due to the absence of functional receptors and progenitor cells. A lack of angiogenesis results in deficient delivery of oxygen and nutrients, further contributing to poor healing.[13]

CHARACTERISTICS AND PATHOPHYSIOLOGY OF TYPES OF WOUNDS

All wounds have the potential to become chronic. Most common chronic wounds can be classified into pressure

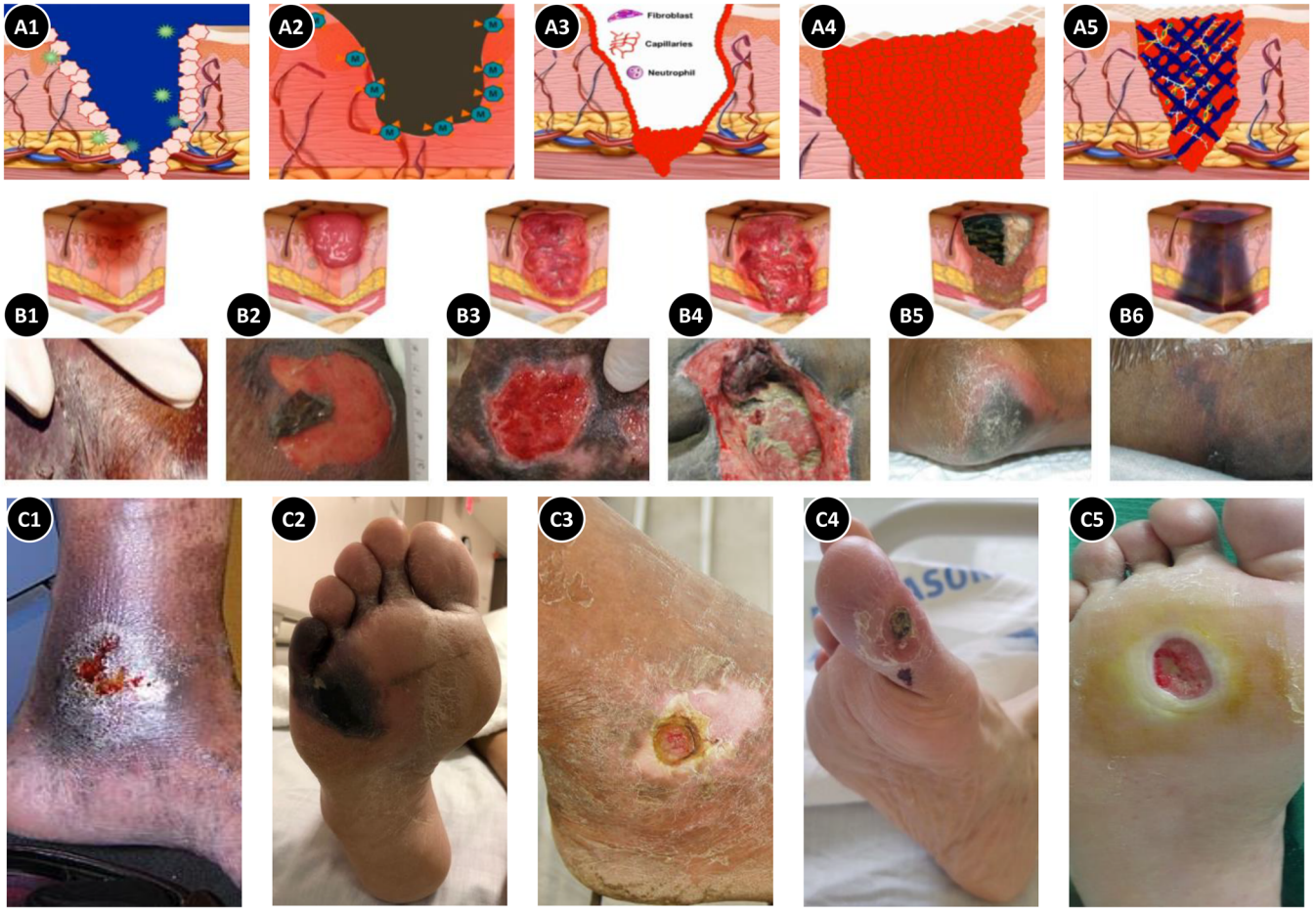

FIGURE 1 Wound care in older adults. (A) Stages of wound healing showing. (A1) hemostasis, where platelets form a clot and release growth factors (1–3 h); (A2) inflammatory phase with neutrophils and macrophages entering wound site to clear dead tissue (1–3 days); (A3) proliferative phase which consists of formation of capillaries and collagen framework by fibroblasts to form granulation tissue (3–21 days); (A4) epithelialization, which is an essential step between granulation and maturation indication wound closure; (A5) maturation, which is the final phase where collagen reorganizes, matures and remodels. (B) NPIAP stages of pressure injury. (B1) Stage 1 pressure injury showing non-blanchable erythema of intact skin; (B2) Stage 2 pressure injury showing partial thickness skin loss with exposed dermis; (B3) Stage 3 pressure injury showing full thickness skin loss; (B4) Stage 4 pressure injury showing full thickness skin loss and tissue; (B5) Unstageable pressure injury showing obscured full thickness skin and tissue loss; (B6) Deep tissue pressure injury showing persistent non-blanchable deep red, maroon, or purple discoloration. (C) Vascular and neuropathic wounds. (C1) Venous ulcer with associated venous stasis dermatitis (web source able to use for educational purposes); (C2) Arterial insufficiency ulcer with gangrene of 5th toe and part of foot; (C3) full thickness arterial insufficiency ulcer on ankle showing characteristic punched out wound with smooth edges; (C4) arterial ulcer on great toe; (C5) Malum perforans ulcer.

Source: (B) Used with permission from the National Pressure Injury Advisory Panel (NPIAP). Copyright 2020 NPIAP. (C) Used with permission from Jonathan Hasson, MD C2 and C4. Unknown web source C1, C2, and C3

injury, vascular (venous and arterial), or diabetic ulcers. Chronic wounds share common features; however, understanding the underlying cause is key to successful management.[14]

Pressure Injury

Pressure injury or ulcers develop as a result of pressure or shear forces to skin overlying bony prominences leading to ischemia/reperfusion and tissue injury.[15] Older skin is much more vulnerable to pressure injury due to multiple risk factors, including immobility, nutritional deficiency, urinary or fecal incontinence, and chronic diseases. Current pressure injury classification is based on the clinical appearance of skin and may not necessarily represent the extent of underlying damage, especially in stage 1 or deep tissue pressure injury.[16]

The term pressure ulcer was replaced by pressure injury by National Pressure Injury Advisory Panel (NPIAP previously NPUAP) consensus in 2016. Pressure injury represents all stages including stage 1 and deep

tissue pressure injury.[17] The pressure injury classification cannot be used for any other type of wounds (Figure 1B).

Vascular wounds

Chronic lower extremity ulcerations occur in up to 5% of the population over 65 years of age[18] and are attributed to venous insufficiency, arterial disease, prolonged pressure, neuropathy or a combination.[19-21] It is estimated that the majority of lower extremity vascular wounds are venous in origin followed by arterial disease, with a quarter of the wounds reflecting mixed etiology with both arterial and venous disease as causative factors.[22-24]

These wounds affect an estimated 2.4–4.5 million people in the United States. They last, on average, 1 year, recur in up to 60–70% of patients, and are a significant cause of morbidity due to loss of function and decreased quality of life.

Venous ulcers

The pathophysiology of venous ulceration is venous reflux that results in increased ambulatory venous pressures leading to superficial chronic inflammation. As this condition, which is associated with aging, progresses, the inflammation and increased hydrostatic pressures lead to the clinical manifestations of chronic venous disease and ulceration.[25] The ulcers are usually shallow and irregularly shaped. Physical findings helpful in determining venous etiology are lower extremity edema, thickening of the skin, discoloration/hemosiderin deposition, large varices, and old healed ulcers[26] (Figure 1C). The most important study to assess venous disease is an ultrasound that includes venous waveform analysis in the superficial and deep systems, color flow analysis, compressibility, waveform analysis, analysis of deep and superficial reflux, and thrombus morphology (if any). Confirmatory studies include catheter-based venography, CT venogram, and/or MR venography examination. The mainstay of treatment of venous wounds is compression (often referred to as UNNA boots), underlying dressings to control/contain drainage, and elevation. For severe venous disease, a vascular surgery referral is helpful. Early treatment of superficial reflux with either radiofrequency or laser endovenous ablation, or ultrasound-guided foam sclerotherapy may speed healing, and decrease recurrence rates.

Arterial insufficiency ulcers

Atherosclerosis of arterial vessel walls manifesting as peripheral arterial disease (PAD) can range from asymptomatic disease to critical limb ischemia.[27] PAD is the foremost cause of wound-related mortality and disability-adjusted life years.[28] In contrast to venous ulcers, arterial insufficiency ulcers are particularly difficult to heal due to low tissue oxygen delivery, decreased trans-capillary diffusion, and local acidosis of the wound bed (Figure 1C). Often arterial and venous insufficiency co-exist challenging the diagnosis and management of lower extremity wounds. Most arterial ulcers are full thickness, punched out with smooth edges (Figure 1C). The initial evaluation for suspected arterial compromise includes measures of ankle brachial index, pulse volume recording, and toe perfusion. However, vascular surgery referral and diagnostic arteriography may be necessary if no objective improvement is seen despite a trial of optimal wound care. With substantive tissue loss and intercurrent infection, early revascularization (open or endovascular) can speed wound closure.

Biopsy of the wound (especially in the presence of long-term chronic ulceration) may be indicated to rule out associated scar carcinoma.

Neuropathic ulcers

Neuropathic ulcers are a serious and potentially fatal complication of diabetes. About 10–15% of diabetic patients develop foot ulcers.[29] The primary pathophysiologic contribution to diabetic wounds is the presence of neuropathy. The lack of sensation leads to foot injuries and deformities, abnormal pressure points, and ultimately to foot ulcers.[30,31] One specific example of diabetic ulcer is the development of both sensory and motor neuropathy, which results in wasting of the dorsal interosseous muscles, leading to splay toe or claw foot deformity. This causes the plantar metatarsal fat pads to be disposed anteriorly, leaving little padding under a metatarsal head. Combined with a sensory defect, this leads to skin erosion and the so-called "malum perforans" ulcer (Figure 1C). Glucose control becomes essential for wound healing in diabetic patients; the goal is to maintain blood glucose at less than 200 mg/dl. Initial treatment is pressure redistribution by offloading the foot with instant or irremovable total contact casting (iCC)[32] and excellent custom footwear. A multidisciplinary approach that includes topical treatments like becaplermin gel was recently reviewed.[31] In some cases, procedures to remove or adjust the metatarsal head may be considered. Limb salvage requires extensive long-term wound care. In severe cases of ulceration, amputation may be the best choice for the patient and should not be regarded as treatment failure.

Atypical wounds

Atypical wounds are a common occurrence in older adults. Atypical wounds or ulcers are those that show unusual clinical features including histology, localization, or resistance to standard therapies. They are generally caused by neoplastic, inflammatory, vasculopathic, hematologic, infectious, or drug-induced etiologies. Examples of atypical wounds are malignant wounds or ones that develop due to vasculitis, gout, autoimmune disorders, calciphylaxis, pyoderma gangrenosum, and trauma as well as bite wounds.[33] Chronic non-healing ulcers that do not respond to appropriate management should raise concern for malignancy and a biopsy should be performed between 4 and 12 weeks after a trial of appropriate treatment.[34]

WOUND ASSESSMENT

Wound assessment not only consists of measuring and monitoring clinical features of the wound, but also recording risk factors and elements important in helping or hindering wound healing. A comprehensive wound assessment includes knowledge of patient co-morbidities, medication use, nutritional status, mobility, and continence. The wound measurement includes noting the size (length, width, depth) as well as wound characteristics such as drainage, odor, presence of slough/necrotic tissue, infection, and condition of the wound bed. There is no standardized method established for wound measurement. Currently, there are various techniques ranging from the simple to the more complex; for example, ruler measurements, acetate tracing, contact/digital planimetry, as well as structured light devices; to measure wounds. Many of these methods would be considered three-dimensional as they measure depth as well as width and length.[35] The same method should be used for monitoring a wound over time to reduce variability in reporting. Particular attention should be given to features such as undermining, tunneling, and wound edges.

GENERAL PRINCIPLES OF WOUND MANAGEMENT

Management of chronic wounds includes both disease-specific approaches as mentioned above, as well as targeted treatments to promote wound healing.[36] To achieve successful closure of a chronic wound, a systematic framework of wound bed preparation[37] is recognized as the best practice for wound healing. It is represented by the acronym TIME (tissue, inflammation/infection, moisture, and edges) (Table 1). In clinical practice, this means preparing the tissue for healing by removing devitalized tissue, controlling excessive inflammation and infection, maintaining moisture balance with appropriate dressing use, and maintaining healthy edges to promote epithelialization.

The concept of wound bed preparation is applicable to potentially healable wounds. For non-healable wounds (where the underlying cause cannot be corrected) or maintenance wounds (with patient/health system challenges-despite correctable cause), a different approach may be needed. This may include periodic aggressive debridement and management of inflammation/infection.[38]

NUTRITION

Compromised nutritional status is fairly common in older adults above the age of 65 years. Inadequate protein intake is known to impair wound healing.[39] Use of protein supplements has been shown to improve the rate of wound repair, particularly in long-term care residents.[40] Correction of vitamin deficiencies like vitamin C and zinc, may lead to improved wound healing.[41]

WOUND TREATMENT

Removal of dead or devitalized tissue with debridement is the first step in promoting the healing of chronic wounds. This can be accomplished using various debridement methods like autolytic, enzymatic, mechanical, surgical, low-frequency ultrasound, or sterile maggots.[42] The exact choice should incorporate patient preference and whether their goal is healing or preventing the wound from getting worse. The decision to debride lower extremity ischemic wounds should be made with extreme caution and a vascular assessment should be performed before debridement. Dry, ischemic lower extremity wounds are best treated conservatively.[43] Debridement prepares the wound bed for the next step, which is the formation of granulation tissue to provide the foundation for new skin formation.

Chronic wounds carry a high antimicrobial burden. Controlling bacterial overgrowth and managing infection is another key to promote wound healing. Wound cleansing can be achieved with normal saline or other commercial wound cleaners. To reduce bacterial bioburden, dressings impregnated with honey (medihoney), iodine, silver, methylene blue, or polyhydroxybutyrate (PHB) can be used. Metronidazole gel is used to decrease the presence of anaerobes, especially in ischemic or fungating wounds.[44,45]

TABLE 1 TIME principle of wound healing

Characteristics	Defect	Intervention needed	Outcome expected
Tissue	Debris, dead or necrotic tissue	Debridement	Restore wound base
Inflammation/ infection	High level of bacteria or inflammation	Antimicrobials and wound cleansing	Reduce inflammation and lower bacterial counts
Moisture balance	Dry, desiccated wound or excess fluid	Appropriate dressing to either provide moisture or absorb excess	Restore cell migration and avoid maceration
Edges	Arrested migration of epithelial cells	Moisture balance, skin protectants, or debridement of edges	Healthy edges to promote epithelial migration

TABLE 2 Common wound dressings based on wound type

Wound appearance	Type of dressing
Necrotic	Hydrocolloid (autolytic debridement) Collagenase (enzymatic debridement) Honey Maggots Ultrasound
Inflammed/ infected	Iodine Silver or sulfa containing creams Polyhexamethylene biguanide Metronidazole 1% Mupirocin 1% Acetic acid 0.5%
Draining	Alginate Absorbent foam Hydrofiber
Non-draining/ dry	Transparent film Hydrocolloid Hydrogel Collagen gel
Advanced	Autograft Allograft Skin substitutes with natural or synthetic extracellular matrix (e.g., Apligraf®, Dermagraft®) Growth factors Gene or cell-based therapies Hyperbaric oxygen therapy Ultrasound Electrostatic stimulation Plasma treatment

Moisture balance is important during wound healing in order for cells like fibroblasts to proliferate, migrate, and synthesize collagen. A lack of moisture will result in a dry wound that hinders cellular migration. Using a gel-based dressing or foam dressing may be more appropriate in this situation. Alternatively, excessive drainage will result in wound maceration with damage to surrounding skin, leading to failure of epithelialization. Use of absorptive dressings like alginates and foams, and in appropriate settings using negative pressure wound therapy (NPWT) avoids maceration. Use of skin protectant before dressing application is a key step in protecting the adjacent skin, especially in frail patients.[46]

Difficult to heal wounds may require advanced dressings if that is within the patient's goals of care. Human-derived skin substitutes suspended in animal or synthetic extracellular matrix and other bioengineered dressings are considered in some cases. However, these dressings require special training and cost may limit their use. (See Table 2 for some commonly used dressings based on wound characteristics.)

In addition to wound treatment, specific attention to the cause of the chronic wound is an integral part of the management strategy and mitigates the risk of recurrence. Pressure injury requires pressure reduction surfaces and techniques, like low air loss or air fluidized mattresses and repositioning. Compression therapy is the mainstay of venous ulcers. Arterial ulcers may need referral to vascular surgery for consideration of revascularization. Diabetic foot ulcers require pressure reduction with iCC.

Despite aggressive wound management, some wounds remain difficult to close, especially atypical wounds like malignancies, rheumatologic wounds, or calciphylaxis. For others, the goal of treatment becomes palliative management, which is a common strategy in debilitated older adults who seek a comfort-focused approach. See Table 2 for some commonly used dressings that can be utilized for the management of chronic wounds or for use in palliative situations. In the latter, where the goal is to keep the older adult comfortable, dressings that require less frequent changes and, at the same time, control the amount of moisture in the wound bed are preferred. Examples include absorbent foam and other soft dressings that can remain in place for up to a week.

Hyperbaric oxygen therapy (HBOT) has many physiologic effects that could promote tissue repair, and reports of its efficacy in wound healing date back to the 1950s.[47] HBOT has multiple approved indications and is used for the treatment of carbon monoxide poisoning,

decompression sickness, and gas embolism. Consequently, HBOT is now available in many urban settings, which has led to its use for a variety of poorly healing wounds for which the data are still controversial. Expert consensus is that the use of HBOT in chronic wounds is best supported in diabetic foot ulcers, but its long-term efficacy even in this setting remains to be established.[48]

Experimental therapies for chronic wounds and interventions that are not widely available (such as 3D bioprinting, gene/growth factor/stem cell-based interventions, electrostatic, plasma, ultrasound, and laser treatments), are evolving and have been recently reviewed by others.[49-51] Broad use of many of these novel methods will be limited by availability, cost, and the necessary expertise for effective utilization.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Wahila Alam wrote the first draft. Jonathan Hasson contributed significantly to vascular wounds section. All coauthors contributed to subsequent versions including the final article.

SPONSOR'S ROLE

None.

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